Historic, archived document

Do not assume content reflects current scientific knowledge, policies, or practices.

LIBRARY
CURRENT SERIAL RECORD
NOV 13 1942
U.S. DEPARTMENT OF AGRICULTURE

Circular No. 628

January 1942 • Washington, D. C.

UNITED STATES DEPARTMENT OF AGRICULTURE

FOWL PARALYSIS

(Avian Leukosis Complex)

By W. J. HALL
Veterinarian, Animal Disease Station
Bureau of Animal Industry

416218°—42

Foreword

Losses to the poultry industry of this country due to diseases have been estimated to run into millions of dollars annually and fowl paralysis has been held to be responsible for half of these losses. The Bureau of Animal Industry undertook active work on this disease at the Beltsville Research Center, Beltsville, Md., in 1931. Studies of fowl paralysis by a number of experiment stations have also been conducted for a considerable period. Owing to the difficulties presented from a research standpoint, an intensive cooperative approach to the problem was begun at the Department's newly established Regional Poultry Research Laboratory at East Lansing, Mich., in 1939. Twenty-five cooperating experiment stations of the North Central and Northeastern States have combined in an effort to solve this problem. The work of the Regional Laboratory, the experiment stations, and the Bureau at Beltsville is a coordinated effort to develop and clarify knowledge concerning the disease. This publication has been prepared to disseminate general information on what is at present known of fowl paralysis.

John R. Mohler, *Chief,*
Bureau of Animal Industry.

Fowl Paralysis

(Avian Leukosis Complex, Lymphomatosis, Range Paralysis)

By W. J. Hall, *veterinarian*
Animal Disease Station, Bureau of Animal Industry

CONTENTS

DISTRIBUTION OF FOWL PARALYSIS

Fowl paralysis and related leukemic diseases (those involving an abnormal number of leucocytes in the blood) are now prevalent practically throughout the world, having been reported from all parts of the United States, and from Canada, England, Germany, France, Italy, Hungary, Norway, Sweden, Denmark, Japan, South America, and South Africa. Fowl paralysis is relatively rare in species of poultry other than the chicken. Turkeys and pheasants are occasionally affected.

Although losses from fowl paralysis have been extensive only during the last decade, the disease was first reported by Marek (*12*)[1] in Hungary in 1907. In the United States, Kaupp (*9*) recognized the disease in 1914, and reported on it in 1921. During the last 15 years numerous investigators in this country and abroad have endeavored to solve the fowl-paralysis problem with respect to cause, method of spread, and control, but with only partial success.

FORMS OF THE DISEASE

Fowl paralysis produces such varying symptoms and tissue changes that it has been called the avian leukosis complex. The more common names, however, are fowl paralysis and range paralysis because of the most prominent symptom, paralysis. Although rather well established in popular usage, the term "paralysis" is inadequate and misleading from a scientific standpoint because, in the restricted sense, it is applicable to only one form of the disease and a symptom which may be seen in other diseases. The following forms of the disease, named after the organs or tissues most prominently affected, are recognized: Nerve form (neural lymphomatosis), eye form (ocular lymphomatosis), visceral form (visceral lymphomatosis), blood form (leukosis), and bone form (osteopetrosis).

[1] Italic numbers in parentheses refer to Liteurature Cited, p. 13.

A particular complication in determining facts concerning the disease has been these multiple manifestations and the difficulty of obtaining, for control purposes, susceptible birds which are free of the disease and which will remain so during the experimental period as a check on the principal groups exposed to the disease.

The nerve form (neural lymphomatosis) is characterized by paralysis of the legs, wings, or neck.

The eye form (ocular lymphomatosis) is characterized by grayness of the iris, and irregularity of the pupil.

The visceral form (visceral lymphomatosis) is characterized by enlargement (tumorlike formation) of the internal organs.

The blood form (leukosis) is characterized by marked changes in the blood, including anemia.

The bone form (osteopetrosis) is characterized by thickening of the long bones, particularly the shanks.

CAUSE

The cause of fowl paralysis is not yet definitely known. However, the disease may be transmitted artificially from bird to bird by inoculation of filtrates obtained from the organs of a diseased bird. Hence, indications are that the causative agent of this disease is a filtrable virus, an extremely small organism which cannot be seen with a microscope, and which is able to pass through the finest bacteria-retaining filter.

Just what role other agents or conditions such as diet, management, parasites, and heredity play in causing the disease is not entirely clear, as these factors have not been fully investigated. Limited experiments in feeding different diets and supplements by Wilcke and coworkers (*17*) did not show diet to be an important factor in the incidence of the disease. Management, particularly with reference to the rearing and segregation of young chicks, is said by some investigators—Kennard and Chamberlin (*10*), Johnston and Wilson (*7*), and Emmel (*2*)—to have a bearing on the incidence of the disease. It is believed by some that heavy parasitism renders fowls more susceptible to fowl paralysis by lowering their resistance, but the disease also occurs in battery-reared chicks which are free of parasites.

There are two theories concerning the causative agent or agents, among investigators of this disease. One is that all forms of the disease are caused by the same agent. The other school of thought believes that the different forms of the disease are caused by two or more distinct though possibly related agents or viruses. Two or more forms of the disease may be present simultaneously in a fowl and they may occur in several combinations. Usually, however, one form of the disease predominates in a flock.

SYMPTOMS AND POST MORTEM OBSERVATIONS

In the nerve form (neural lymphomatosis) of the disease the legs, wings, or neck may lose their power of movement in varying degrees. At the onset of the disease the symptoms may be slight and scarcely noticeable, such as a slight limp or weaving gait, a partial dropping of the wing, or the head may be held at an unusual angle. Later the leg or wing may lose more and more of its usefulness until the bird is unable to stand, or it may drag one or both wings on the ground.

A characteristic posture in an advanced case of paralysis is one in which the bird lies on the side of the breast with one leg extended forward and the other backward, as shown on the cover page. In other instances torticollis, or wry neck, may be the most prominent symptom, in which case the head may be drawn to one side, tilted upward and backward, or held down near the ground with the beak pointing up. Occasionally, the breathing apparatus is involved causing the bird to gasp for air. This symptom may closely resemble the inspiratory gasps seen in the respiratory diseases such as infectious laryngotracheitis or infectious bronchitis, but in respiratory paralysis

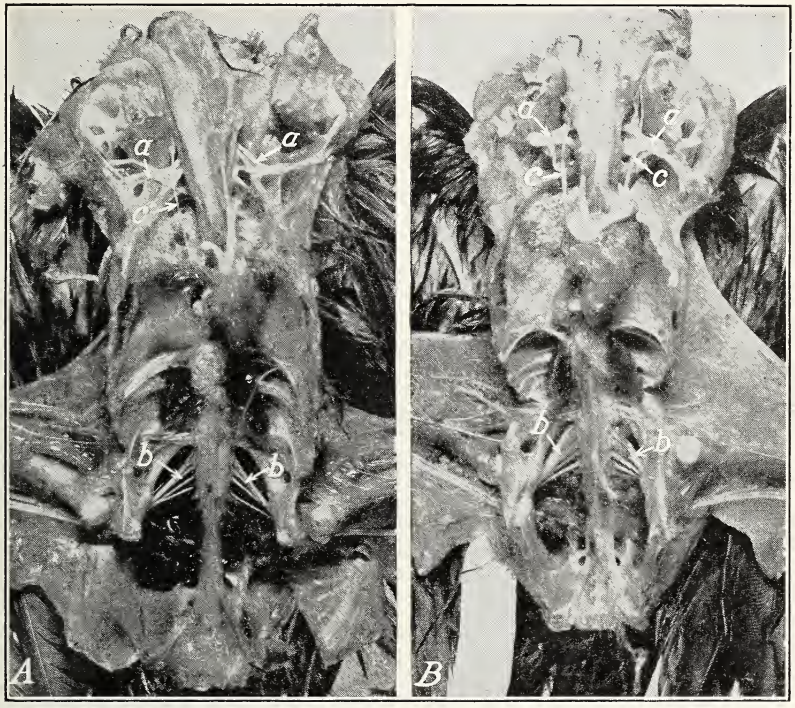

FIGURE 1.—*A*, Carcass of a fowl showing normal nerves (left vagus nerve not shown); *B*, carcass of a fowl dead of paralysis; *aa*, brachial nerves to wings; *bb* sciatic nerves to legs; *cc*, vagus nerves to heart, lungs, and digestive tract. Note swollen nerves in carcass of fowl dead of paralysis.

there are no exudates and no breathing noises. The function of the digestive tract is frequently disturbed, especially in the prolonged cases, as evidenced by extreme loss of flesh, and diarrhea or constipation. The appetite, however, usually remains good and the bird will eat so long as it is able to reach food.

Post mortem examination shows little or no change in the internal organs in acute cases other than enlargement of the nerve (fig. 1, *A* and *B*) supplying the affected limb. This enlargement of the nerves supplying the wings or legs (fig. 1*A*, *a* and *b*) is more often seen in the portion of the nerve where it emerges from the spinal column. In the case of the nerve (sciatic) supplying the legs, it is necessary to

remove the overlying middle lobe of the kidney to observe this portion of the nerve. The nerves most often affected in this form of the disease are the brachial (supplying the wing), the sciatic (supplying the legs), and the vagus (supplying the heart, lungs, and digestive tract) (fig. 1).

In the eye form of the disease, ocular lymphomatosis, the most prominent symptoms are loss of pigment from the iris, and changes in the pupil. This manifestation of the disease is often called gray eye, glass eye, pearly eye, or fish eye. Grayness of the iris is caused by replacement of the normal orange or bay pigment by a type of immature, white blood cell. This fading of the iris pigment usually

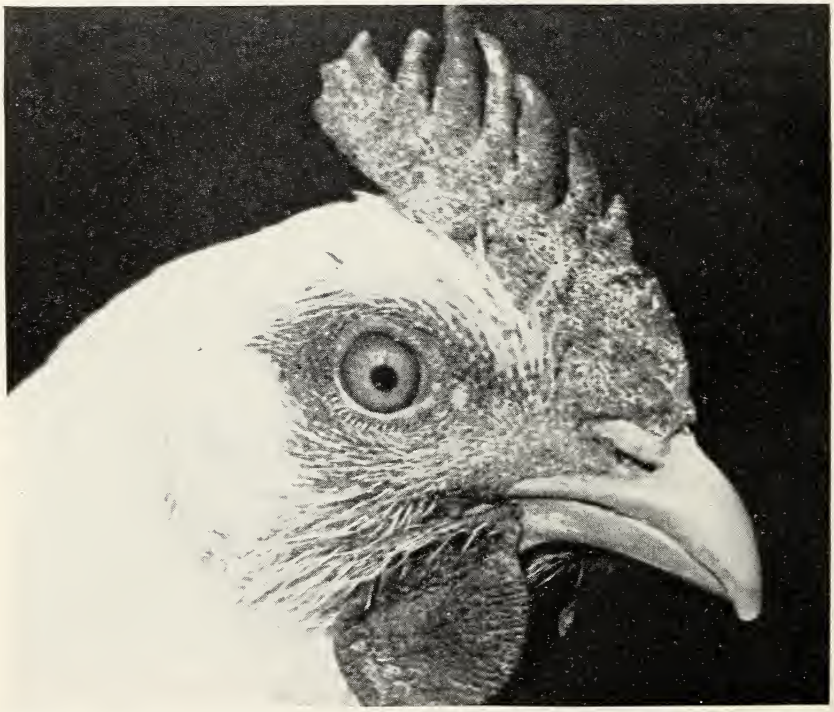

FIGURE 2.—A fowl affected with lymphomatosis, ocular form. Note gray iris.

begins at the inner edge surrounding the pupil and gradually progresses until the iris becomes completely gray, or bluish-gray. In rare instances it becomes dark brown. Usually along with the gray iris (fig 2) the pupil becomes affected, either contracting to pinhead size or becoming abnormally dilated. Usually in such cases the power of light accommodation is lost. In other cases the pupil becomes irregular, ragged, or eccentric. These changes in the eye result in partial to complete loss of sight.

In the visceral form of the disease, visceral lymphomatosis, there may be no visible symptoms, especially if the tumors are confined to the internal organs. In cases of long standing, however, emaciation and depression may be noted, and occasionally a swollen, dropsical

abdomen may be observed. If the tumors are external they may be seen on any part of the body, but they frequently develop in the feather follicles, and vary in size from about ⅛ inch to more than 1 inch in diameter (fig. 3). Frequently the centers of the large skin tumors

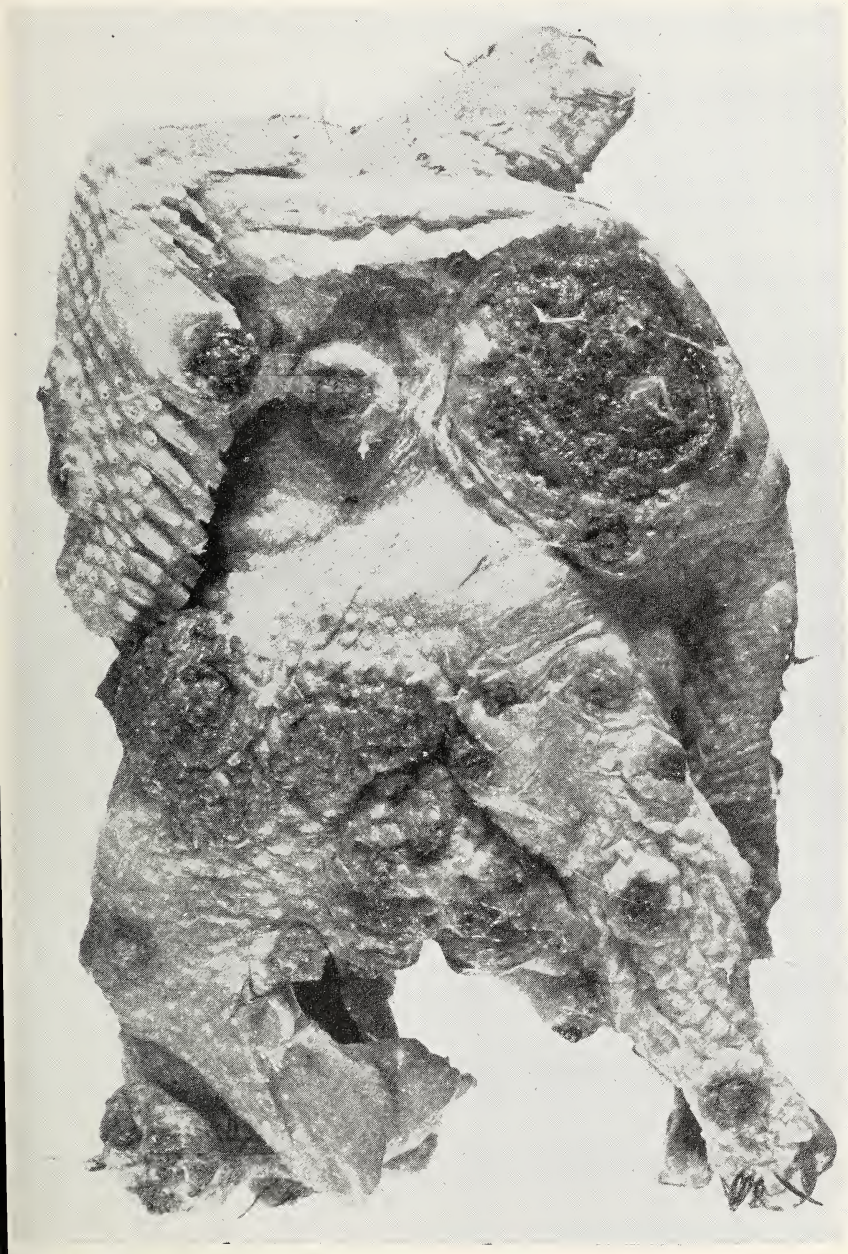

FIGURE 3.—Lymphomatosis of skin. Carcass of chicken showing multiple skin tumors which have ulcerated and become encrusted with scabs.

break down and become ulcerated. Tumors may be found in any of the internal organs but most frequently in the liver, ovary, kidneys, spleen, and less often in the heart, lungs, and digestive tract. These so-called tumors are the result of proliferation in and infiltration of the organ with enormous numbers of an immature type of white blood cell causing enlargement of the organ and interference with its func-

FIGURE 4.—Visceral lymphomatosis. Livers from chickens affected with so-called "big-liver disease." Nodular lymphoid cell infiltration of: *A*, The liver; *B*, the spleen; *C*, diffuse lymphoid cell infiltration of the liver. Compare with normal: *D*, Liver; *E*, spleen.

tion to a greater or less extent. The tumors are white to grayish pink in color, and of a fleshlike consistency. In some cases there is a uniform, diffuse infiltration of the organ by the tumor cells giving it a grayish-red appearance, while in other cases the tumor cells localize in the organ in the form of gray or white nodules interspersed with normal tissue (fig. 4). In this form of the disease the internal organs may become enormously enlarged by the white-cell infiltration; the liver sometimes weighs 1 pound or more, giving rise to the term "big-liver disease." The ovary is occasionally enlarged to the size of a billiard ball.

The blood form of the disease, leukosis, is usually accompanied by anemia, as indicated by pallor of the head and general unthriftiness.

The blood is frequently thin and watery in appearance. On post mortem examination there is usually enlargement of the liver and spleen, and these organs are a brighter red than normal. Pin-point gray spots may be seen occasionally in the livers of young chickens. The kidneys may be either paler than normal or swollen and reddish. Small hemorrhages may sometimes be noted on the heart and intestines. There is also an extensive loss of flesh in many cases. In this form of the disease a great increase in the number of white blood (lymphoid type) cells may be observed on microscopic examination of the blood.

The bone form of the disease, osteopetrosis, as described by Jungherr and Landauer (*8*), occurs but rarely. It consists in a thickening of the long bones and is especially noticeable in the shanks which are most frequently affected. This thickening of the bones appears to take place both from the inside, or marrow cavity, and on the outside. Deposition of bone may continue until the marrow cavity is nearly obliterated and the bone loses its natural shape. The shanks often become thickened, rough, and bulged, giving the bird a stilted appearance. The joints are not affected. The relationship of this form of the disease to fowl paralysis is not yet well established.

INCUBATION PERIOD AND COURSE

Under ordinary conditions the length of time between exposure of birds to the causative agent of this group of diseases and the first appearance of symptoms, known as the incubation period, appears to be approximately 6 weeks in the nerve form of the disease, probably depending on the virulence of the causative agent and the resistance of the birds. In the blood form of the disease the incubation period following artificial inoculation may be as short as 1 to 2 weeks. Also in this form of the disease it has been demonstrated by Hall, Bean, and Pollard [2] and Jarmai (*5*) that the incubation period and course may be materially shortened by continuous serial passage of the infective agent through young chicks.

The course (the time between the appearance of symptoms and death) of the nerve form of the disease may be rapid in acute cases in young birds. In some cases it may be only a few days from the time the first limp is noticed until the bird is prostrate or dead. On the other hand, both the nerve and eye forms of the disease often become chronic, especially in older birds, and they may linger on for weeks or months. It is not unusual for fowls with gray eye to continue laying for a considerable time. Disease changes in the eye do not become prominent until the birds are about 4 months of age or older. In the visceral form of the disease the length of the incubation period and the course are uncertain, as in this form of the disease no symptoms may be noticeable until just before death.

After paralysis appears in a flock the disease spreads slowly, only a bird or two coming down at a time, but new cases keep developing so that losses may be high over a period of a year.

---

[2] Unpublished data.

DISSEMINATION OF THE DISEASE

Considerable evidence from both experimental work and field observations indicates that the disease, particularly lymphomatosis, is spread by contact with infected chickens and by contact with pens which have been contaminated by infected birds, as shown by Lee and coworkers (*11*), Thorp and Graham (*16*), Kennard and Chamberlin (*10*), and Gildow et al. (*3*). The blood form of the disease (leukosis) appears to be less contagious, and it is suggested by Olson (*13*) that it may originate spontaneously.

The role of the egg in transmission of this group of diseases has not been determined. Observations by research workers as well as by poultrymen indicate that susceptibility or resistance may be inherited, but it is not indisputably established that the disease agent is actually passed through the egg, although the work of Durant and McDougle (*1*) lends support to this view. Brandly and Cottral [3] of the Regional Poultry Research Laboratory at East Lansing, Mich., and Hall and others (*4*) at Beltsville, Md., have demonstrated that the leukosis agent may be propagated in the developing egg by intravenous inoculation of the chick embryo.

Other possible agencies which may be involved in the spread of the disease include attendants, parasites, wild birds, and vermin. Whether or not the incubator is a factor in the spread of the disease has not been investigated. There is some evidence that the attendant may carry the disease from infected fowls to susceptible young chicks.

The role of mosquitoes and mites in the spread of the leukemialike diseases has been investigated to a limited extent as reported by Ratcliffe and Stubbs (*15*) and Johnson (*6*) but results of these experiments were conflicting.

Results of an investigation by Johnson (*6*) of the transmission of this group of diseases in the process of pox vaccination indicated this to be a possibility when the follicle brush method is used. However, these results have not been confirmed.

SUSCEPTIBILITY

Newly hatched chicks are most susceptible to the disease, according to Gildow and others (*3*). Resistance increases with age until maturity, at which time the bird is very resistant to ordinary means of natural exposure, as shown by Pappenheimer and coworkers (*14*). Under natural conditions chickens from a few weeks old to a year of age or older become affected. The greatest number become affected, however, between 4 and 10 months of age. Whether this is on account of the variation in individual resistance, the strain of beginning egg production, or for other reasons is not clear. There are no marked breed or sex differences in susceptibility. However, differences in susceptibility of different strains and families of fowls, as well as individual differences, have been observed.

---

[3] Personal communication.

LOSSES FROM FOWL PARALYSIS

The mortality from fowl paralysis may range from 10 to more than 50 percent of the birds in the flock. Only in rare cases does a bird recover completely after the appearance of clinical symptoms. Rather heavy losses may occur in a flock for 3 or 4 years in succession following an outbreak after which the losses usually subside somewhat, probably as a result of survival of the most resistant birds or a decrease in virulence of the causative agent. In some outbreaks, however, losses continue year after year with little decrease.

In affected birds egg production is either suspended altogether or is greatly decreased. In cases of the gray-eye form of the disease, however, production may be maintained for a considerable time. Birds with the tumor type of the disease also may continue production for a time, even with a tumor in the ovary.

METHODS OF CONTROL

Treatment.—No drugs, tonics, medicines, vaccines, bacterins, or special feeds of any kind have been found effective in preventing or curing the disease. The feeding of lettuce in large quantities and also the feeding or injection of wheat-germ oil have been advocated for the cure of the disease, but carefully controlled tests of these remedies by poultry pathologists have not shown them to be of any value.

Prevention.—Since so little is known of the cause, methods of diagnosis in the live bird, and methods of dissemination, no control measures can be recommended which will be completely satisfactory. Sanitation and breeding for resistance appear to offer the best means of control in the light of present knowledge. Sanitary measures which are recommended as aids in the prevention of infection are as follows:

Exclude all visitors from poultry premises.

Avoid, so far as possible, bringing in new stock such as hatching eggs, baby chicks, pullets, or cockerels from outside sources. If breeding males must be purchased, get mature birds (yearlings or 2-year-olds) which are in good health and from a healthy flock. However, it should be realized that bringing in any outside stock is dangerous because of the difficulty of diagnosis of this group of diseases in live birds. It is advisable to hatch eggs from one's own flock of 2-year-old hens or older, and use one's own incubator.

Brood all chicks as far removed as possible from the mature fowls. It is advisable also to provide different attendants for the young chicks and for the mature fowls.

Keep the brooder house clean, dry, and well ventilated. Avoid overheating and overcrowding. Let chicks outdoors at an early age.

Rear chicks on a clean grass range which has not been occupied by chickens during the preceding year, or preferably, put them on new ground.

Provide chicks with an adequate, well-balanced diet and clean, fresh water.

Avoid bringing back any birds which have been sent off the premises.

Keep the flock from mingling with neighboring flocks.

Endeavor to get poultry feed in clean, new containers.

All returned crates and containers which might have been contaminated by other poultry should be cleaned and disinfected before being brought into the poultry buildings.

PROCEDURE DURING AN OUTBREAK

The following procedures are recommended for the control of outbreaks of fowl paralysis:

Dispose of all birds showing symptoms of any form of fowl paralysis such as leg or wing lameness, incoordination (weaving or staggering gait), wry neck, diseased eyes including gray irises of any degree, irregular, ragged, or eccentric pupil and bulging eyeballs, tumors on the skin, emaciation (extreme loss of flesh), pale heads, flaccid, pendulous crops, gaspy breathing, and swollen, dropsical abdomen.

It should be borne in mind that all diseased birds cannot be removed at one or two cullings as this group of diseases develops gradually over a period of months. Furthermore, some hens may have developed internal tumors while still in production and without showing noticeable signs of disease. The elimination of diseased birds should, therefore, be a continuing practice so that each bird is removed from the flock as soon as it shows visible signs of disease. In this way the reservoir of infection is diminished and unprofitable birds are eliminated.

At the end of the laying or breeding season the question of replacement or replenishment of the flock arises. Should the old flock be disposed of entirely and new stock, baby chicks, or pullets, purchased, or should replacements be made by breeding the healthy survivors of the old flock? The consensus of opinion among investigators of this group of diseases now favors the latter course as the most promising control measure. However, this procedure cannot be expected to give immediate relief from losses, as developing resistance to disease by breeding is a gradual process which must be built up by careful selection of the most resistant individuals and families over a period of years.

If, on the other hand, it is decided to dispose of the entire flock after a paralysis outbreak, and bring in new stock, no assurance can be given that the new stock, whether baby chicks or laying fowls, will be free from the disease unless they are obtained from a flock of proved resistance, and such flocks are at present hard to find.

In any event, replacement chicks should be reared in isolation and managed as suggested in the discussion of prevention.

If the new crop of pullets is to be housed in the old laying house it is advisable to remove the old birds and clean the house and yards thoroughly. In cleaning the laying house all dust should be removed from walls and ledges. Remove all litter and hard crusts from the floor and roosts by soaking and scraping. Then disinfect the floors, walls, and ceiling of the house by spraying with a 2½-percent lye solution (1 pound of lye to 5 gallons of water) or a 4-percent cresylic disinfectant. All trash and litter should be raked from the yards and burned. Chickens should be prevented from getting under the laying house or in any place that cannot be cleaned. All feeding and watering equipment and utensils should be thoroughly scrubbed and disinfected and the yards cleaned and well drained so that the sun

can exert its germ-killing power. In the larger yards cultivation of the soil and the growing of crops help to destroy disease-producing agents.

LITERATURE CITED

(1) Durant, A. J., and McDougle, H. C.
1939. studies on the origin and transmission of fowl paralysis (neurolymphomatosis) by blood inoculation. Mo. Agr. Expt. Sta. Res. Bul. 304, 23 pp., illus.

(2) Emmel, M. W.
1939. hemocytoblastosis in the chicken. World's Poultry Cong. Proc., pp. 298–300.

(3) Gildow, E. M., Williams, J. K., and Lampman, C. E.
1940. the transmission of and resistance to fowl paralysis (lymphomatosis). Idaho Agr. Expt. Sta. Bul. 235, 22 pp., illus.

(4) Hall, W. J., Bean, C. W., and Pollard, Morris.
1940. preliminary report on the propagation of the fowl-leucosis virus on chick embryos by intravenous inoculation. Jour. Amer. Vet. Med. Assoc. 97 : 247.

(5) Jarmai, Karl.
1938. Über die wirksamkeit der eiweissfraktionen bei der übertragbaren hühnerleukose. Arch. f. Wiss. u. Prakt. Tierheilk. 73 : [295]–300.

(6) Johnson, E. P.
1937. transmission of fowl leukosis. Poultry Sci. 16 : 255–260, illus.

(7) Johnston, J. W., and Wilson, J. E.
1939. observations on the etiology and control of fowl paralysis. Vet. Jour. 95 : 474–485.

(8) Jungherr, Erwin, and Landauer, Walter.
1938. studies on fowl paralysis. 3. a condition resembling osteopetrosis (marble bone) in the common fowl. Conn. (Storrs) Agr. Expt. Sta. Bul. 222, 34 pp., illus.

(9) Kaupp, B. F.
1921. paralysis of the domestic fowl. Amer. Assoc. Instr. and Invest. Poultry Husb. Jour. 7 : 25–31, illus.

(10) Kennard, D. C., and Chamberlin, V. D.
1936. livability and production of pullet layers may be predetermined. Ohio Agr. Expt. Sta. Bimo. Bul. 21 (183) : 136–140.

(11) Lee, C. D., Wilcke, H. L., Murray, Chas., and Henderson, E. W.
1937. fowl leukosis. Amer. Vet. Med. Assoc. Jour. 91 : 146–162.

(12) Marek, J.
1907. multiple nervenentzündung (polyneuritis) bei hühnern. Deut. Tierärztl. Wchnschr. 15 : [417]–421, illus.

(13) Olson, Carl, Jr.
1940. transmissible fowl leukosis. a review of the literature. Mass. Agr. Expt. Sta. Bul. 370, 48 pp.

(14) Pappenheimer, Alwin M., Dunn, Leslie C., and Cone, Vernon.
1926. a study of fowl paralysis (neuro-lymphomatosis gallinarum). Conn. (Storrs) Agr. Expt. Sta. Bul. 143, [183]–290, illus.

(15) Ratcliffe, Herbert, and Stubbs., E. L.
1935. attempts to transmit chicken leukosis by mosquitoes and by mites. Jour. Infect. Dis. 56 : [301–]304.

(16) Thorp, Frank, Jr., and Graham, Robert.
1936. transmission studies in leucemia. Vet. Med. 31 : 82–85, illus.

(17) Wilcke, H. L., Patterson, F. D., Henderson, E. W., and Murray, Charles.
1933. the effect of the ration upon the incidence of so-called range paralysis. Poultry Sci. 12 : 226–232.

ORGANIZATION OF THE UNITED STATES DEPARTMENT OF AGRICULTURE WHEN THIS PUBLICATION WAS EITHER FIRST PRINTED OR LAST REVISED

Secretary of Agriculture	CLAUDE R. WICKARD.
Under Secretary	PAUL H. APPLEBY.
Assistant Secretary	GROVER B. HILL.
Chief, Bureau of Agricultural Economics	HOWARD R. TOLLEY.
Director of Agricultural Defense Relations	M. CLIFFORD TOWNSEND.
Director of Extension Work	M. L. WILSON.
Director of Finance	W. A. JUMP.
Director of Foreign Agricultural Relations	L. A. WHEELER.
Director of Information	MORSE SALISBURY.
Director of Personnel	T. ROY REID.
Land Use Coordinator	MILTON S. EISENHOWER.
Librarian	RALPH R. SHAW.
Solicitor	MASTIN G. WHITE.
Chief, Office of Civilian Conservation Corps Activities.	FRED MORRELL.
Chief, Office of Plant and Operations	ARTHUR B. THATCHER.
Administrator of Agricultural Marketing	ROY F. HENDRICKSON.
Administrator, Surplus Marketing Administration.	E. W. GAUMNITZ.
Chief, Commodity Exchange Administration.	JOSEPH M. MEHL.
Chief, Agricultural Marketing Service	CLARENCE W. KITCHEN.
Administrator of Agricultural Adjustment and Conservation.	R. M. EVANS.
Administrator, Agricultural Adjustment Administration.	FRED S. WALLACE.
Chief, Soil Conservation Service	HUGH H. BENNETT.
Manager, Federal Crop Insurance Corporation.	LEROY K. SMITH.
Chief, Sugar Division	JOSHUA BERNHARDT.
Administrator of Agricultural Research	E. C. AUCHTER.
Chief, Bureau of Animal Industry	JOHN R. MOHLER.
Chief, Bureau of Agricultural Chemistry and Engineering.	HENRY G. KNIGHT.
Chief, Bureau of Dairy Industry	OLLIE E. REED.
Chief, Bureau of Entomology and Plant Quarantine.	P. N. ANNAND.
Chief, Office of Experiment Stations	JAMES T. JARDINE.
Chief, Bureau of Plant Industry	E. C. AUCHTER.
Chief, Bureau of Home Economics	LOUISE STANLEY.
President, Commodity Credit Corporation	J. B. HUTSON.
Administrator of Farm Security Administration.	C. B. BALDWIN.
Governor of Farm Credit Administration	ALBERT G. BLACK.
Chief, Forest Service	EARLE H. CLAPP, *Acting.*
Administrator, Rural Electrification Administration.	HARRY SLATTERY.

This bulletin is a contribution from

Bureau of Animal Industry ..... JOHN R. MOHLER, *Chief.*

U. S. GOVERNMENT PRINTING OFFICE: 1942

For sale by the Superintendent of Documents, Washington, D. C. - - - - - - - Price 5 cents